Theor Appl Genet (2004) 109:210–214
DOI 10.1007/s00122-004-1619-7

ORIGINAL PAPER

Ch. Singrün · S. L. K. Hsam · F. J. Zeller · G. Wenzel · V. Mohler

Localization of a novel recessive powdery mildew resistance gene from common wheat line RD30 in the terminal region of chromosome 7AL

Received: 10 October 2003 / Accepted: 26 January 2004 / Published online: 10 March 2004

Abstract Segregation analysis of resistance to powdery mildew in a F_2 progeny from the cross Chinese Spring (CS) × TA2682c revealed the inheritance of a dominant and a recessive powdery mildew resistance gene. Selfing of susceptible F_2 individuals allowed the establishment of a mapping population segregating exclusively for the recessive resistance gene. The extracted resistant derivative showing full resistance to each of 11 wheat powdery mildew isolates was designated RD30. Amplified fragment length polymorphism (AFLP) analysis of bulked segregants from F_3s showing the homozygous susceptible and resistant phenotypes revealed an AFLP marker that was associated with the recessive resistance gene in repulsion phase. Following the assignment of this AFLP marker to wheat chromosome 7A by means of CS nullitetrasomics, an inspection of simple sequence repeat (SSR) loci evenly spaced along chromosome 7A showed that the recessive resistance gene maps to the distal region of chromosome 7AL. On the basis of its close linkage to the *Pm1* locus, as inferred from connecting partial genetic maps of 7AL of populations CS × TA2682c and CS × Virest (*Pm1e*), and its unique disease response pattern, the recessive resistance gene in RD30 was considered to be novel and tentatively designated *mlRD30*.

Communicated by C. Möllers

C. Singrün · G. Wenzel · V. Mohler (✉)
Chair of Agronomy and Plant Breeding,
Department of Plant Sciences,
Center for Life and Food Sciences Weihenstephan,
Technical University Munich,
Am Hochanger 2, 85350 Freising, Germany
e-mail: mohler@wzw.tum.de
Fax: +49-8161-715173

S. L. K. Hsam · F. J. Zeller
Division of Plant Breeding and Applied Genetics,
Department of Plant Sciences,
Center for Life and Food Sciences Weihenstephan,
Technical University Munich,
Lange Point 51, 85350 Freising, Germany

Introduction

Wheat is one of the most commonly cultivated crop plants worldwide. Powdery mildew disease caused by the fungus *Blumeria graminis* f. sp. *tritici* (Bgt) is a major threat to wheat production, especially in areas with a cool or maritime climate. The deployment of resistant cultivars is the most economical and environmentally safe means for controlling this disease. However, cultivars with single resistance genes become susceptible shortly after their commercial exploitation due to frequent changes in the pathogen populations (Szunics et al. 1999). One strategy that has been applied to achieve more comprehensive protection is the pyramiding of multiple simply inherited resistance genes or, in other terms, their combination with QRL (quantitative resistance loci, Young 1996) for a given pathosystem in a single genotype. Multiple changes in the pathogen would then be required to render the acquired resistance ineffective. Successful examples of marker-aided pyramiding of major wheat powdery mildew resistance genes have been reported by Liu et al. (2000) and Wenzel et al. (2000).

To date, 32 major gene loci conferring resistance to wheat powdery mildew have been detected, mapped to specific chromosomes, and given official designations (*Pm1-Pm32*, McIntosh et al. 1998, 2003; Hsam et al. 2003). The majority of the wheat powdery mildew resistance genes show dominant inheritance, and only three recessive gene loci—*Pm5* (McIntosh et al. 1967; Lebsock and Briggle 1974), *Pm9* (Favret 1979, cited in McIntosh et al. 1998), and *Pm26* (Rong et al. 2000)—are known so far. Similarly, a small number of recessive genes have also been found to be involved in resistance reactions against leaf rust (*Lr30*, Dyck and Kerber 1981; *Lr48*, Saini et al. 2002), stem rust (*Sr2*, Knott 1968; *Sr12*, Sheen and Snyder 1964; *Sr17*, McIntosh et al. 1967), and stripe rust (*Yr2*, Lupton and Macer 1962).

Recently identified powdery mildew resistance genes and those frequently used in practical breeding have been mapped with molecular markers (Huang et al. 2003). Molecular mapping became the overriding method for

assigning genes to chromosomes. The purpose of the investigation reported here was to identify a dominant and a recessive powdery mildew resistance gene in common wheat line TA2682c. Following establishment as a single gene differential, molecular mapping assigned the recessive powdery mildew resistance gene from the extracted resistant derivative RD30 to the distal region of wheat chromosome 7AL. The position of the recessive resistance gene *mlRD30* relative to *Pm1* was determined.

Materials and methods

Plant materials

Common wheat line TA2682c, which was provided by breeding company Saatzuchtgesellschaft Streng's Erben, Germany, shows broad resistance to powdery mildew. The segregation for resistance to powdery mildew within a F_2 progeny derived from the cross Chinese Spring (CS) × TA2682c approximated a ratio of 13:3, indicating that TA2682c possesses a dominant and a recessive resistance gene. To produce a mapping population segregating for the recessive resistance gene only, we selfed the susceptible F_2 plants. For bulked segregant analysis (Michelmore et al. 1991), two DNA bulks were established by combining equal amounts of DNA from eight to ten resistant individuals collected each from seven segregating F_3 families and ten homozygous-susceptible F_3 families, respectively. Randomly chosen resistant and susceptible F_3 plants were selfed to form a mapping population consisting of 60 F_4 families. The resistant derivatives from F_4 family no. 30 (homozygous for the resistance gene, but not uniform for the genetic background) were designated RD30. Nulli-tetrasomics of CS, originally obtained from the late Dr. E.R. Sears (University of Missouri, USA), were used for chromosome assignment of amplified fragment length polymorphism (AFLP) markers detected in repulsion phase. Lines/cultivars Axminster (*Pm1a*), Ulka (*Pm2*), Asosan (*Pm3a*), Khapli/8*Chancellor (*Pm4a*), Hope (*Pm5a*), Disponent (*Pm8*), Pompe (*Pm1a+9*) and Normandie (*Pm1+2+9*) were used to compare resistance reactions relative to lines TA2682c and RD30.

Evaluation of resistance reactions

The Bgt isolates used for the differentiation of documented major resistance genes were collected from different parts of Europe and selected from single-spore progeny. These are classified under Weihenstephan accession numbers and maintained at the Division of Plant Breeding and Applied Genetics, Technical University Munich. Powdery mildew resistance reactions were surveyed on agar-detached primary leaf segments. The methods of inoculation, conditions of incubation, and disease assessment were according to Hsam and Zeller (1997). Generally, three main classes of host reactions were distinguished: r = resistant (0–20% infection relative to cv. Kanzler), i = intermediate (30–50% infection), s = susceptible (more than 50% infection). To follow powdery mildew segregation, we utilized Bgt isolates for which the resistance genes under investigation confer full resistance (0% infection relative to cv. Kanzler). The following four were chosen: no. 2 is virulent to *Pm2*, *Pm4a*, and *Pm5a*, but avirulent to *Pm1a*, *Pm3a*, and *Pm8*; no. 9 is virulent to *Pm1a*, *Pm2*, and *Pm5a*, but avirulent to *Pm3a*, *Pm4a*, and *Pm8*; no. 10 shows virulence to *Pm4a* and *Pm8*, but avirulence to *Pm1a*, *Pm2*, *Pm3a*, and *Pm5a*; no. 13 is virulent to *Pm1a*, *Pm2*, *Pm4a*, *Pm5a*, and *Pm8*, but avirulent to *Pm3a*. Offspring having an infection level greater than 0% were classified as susceptible. Chi-square tests for goodness-of-fit were used to test for the deviation of observed data from theoretically expected segregation.

Table 1 Description of SSR markers

Locus	Annealing temperature (°C)	Fragment size (bp) in:	
		Chinese Spring	TA2682c
Xgwm350-7AS	55	150	146
Xgwm573-7AS	50	215	201
Xgwm260-7AS	55	160	176
Xgwm63-7AL	55	269	269
Xgwm282-7AL	55	219	187
Xgwm332-7AL	55	234	Null
Xgwm344-7AL	55	133	131

Molecular marker and mapping techniques

Wheat primary leaf tissue was used for DNA extraction essentially following the procedure of Huang et al. (2000a). *Eco*RI+ANN/*Mse*I+CNN AFLPs were generated according to Schwarz et al. (2000). AFLP marker designations were based on the primer combination used and the fragment size estimated with reference to the internal lane size standard GeneScan-500 ROX (Applied Biosystems, Foster City, Calif.). Codes for AFLP primers can be viewed in the standard list for AFLP primer nomenclature (http://wheat.pw.usda.gov/ggpages/keygeneAFLPs.html). Detected loci were marked with an 'X', the basic symbol for molecular marker loci of an unknown function in wheat. PCR protocols for wheat simple sequence repeat (SSR) markers (Table 1) were as described in Röder et al. (1998). Both molecular marker types were detected on an ABI PRISM 377 platform (Applied Biosystems). Fragment size-calling was performed with GENESCAN analysis software ver. 3.0, allele typing with GENOTYPER DNA fragment analysis software ver. 2.0 (Applied Biosystems). Primer sequences for marker *Xsts638* have been reported by Neu et al. (2002). Restriction fragment length polymorphism (RFLP) analysis of marker loci *Xksuh9*, *Xmwg2062*, *Xrz508*, and *Xwg341*, which are described in the Graingenes database (http://wheat.pw.usda.gov/) followed standard methods. Aliquots of 15 μg of genomic DNA from the parental lines were digested with restriction endonucleases *Bam*HI, *Dra*I, *Eco*RI, *Eco*RV, *Hind*III, and *Xba*I. RFLP probes *Xrz508* and *Xwg341* were kindly provided by the Cornell Research Foundation (Cornell University, USA), and *Xksuh9* was provided by Prof. B.S. Gill (Kansas State University, USA). Partial linkage maps were constructed with the computer program JOINMAP 3.0 (Van Ooijen and Voorrips 2002) using the Haldane mapping function. Charts of genetic linkage maps were drawn with MAPCHART 2.1 software (Voorrips 2002).

Results and discussion

Inheritance of powdery mildew resistance from common wheat line TA2682c

Common wheat line TA2682c showed full resistance to each of the 11 Bgt isolates from the standard assortment (Table 2). Monosomic analysis showed a segregation ratio of 13 resistant:3 susceptible (data not shown), indicating the inheritance of a dominant and a recessive resistance gene. This segregation ratio was verified when Bgt isolate nos. 2 and 13 were used on an additional 440 F_2 plants from cross CS × TA2682c (isolate 2: 352 resistant:88 susceptible, $\chi^2_{13:3}$=0.4, P=0.53; isolate 13: 356 resistant:84 susceptible, $\chi^2_{13:3}$=0.02, P=0.89). The degree of misclassification with the two different Bgt isolates was only 0.9%. Only the F_2 population from cross CS monosomic-

Table 2 Seedling reactions of ten wheat cultivars/lines carrying different powdery mildew resistance genes after inoculation with 11 isolates of *Blumeria graminis* f. sp. *tritici*

Line/cultivar	*Blumeria graminis tritici* isolate no.											*Gene*
	2	5	6	9	10	12	13	14	15	16	17	
Axminster/8*Cc[a]	r[b]	r	r	s	r	s	s	s	r	s	s	*Pm1a*
Ulka	s	r	r	s	r	s	s	s	r	s	s	*Pm2*
Asosan/8*Cc	r	s	r	r	r	s	r	r	s	s	r	*Pm3a*
Khapli/8*Cc	s	r	s	r	i	r	s	s	i	s	i	*Pm4a*
Hope	i	s	s	s	r	s	s	r	s	s	s	*Pm5a*
Disponent	r	s	s	r	s	r	s	s	s	s	r	*Pm8*
Pompe	r	r	r	i/s	r	s	s	s	r	s	i/s	*Pm1+9*
Normandie	r	r	r	s	r	s	s	s	r	i/s	i	*Pm1+2+9*
TA2682c	r	r	r	r	r	r	r	r	r	r	r	*Pm3+mlRD30*
RD30	r	r	r	r	r	r	r	r	r	r	r	*mlRD30*

[a] Seven times backcrossed to cv. Chancellor
[b] r, Resistant; i, intermediate; s, susceptible

1A × TA2682c clearly showed a significant deviation from the expected segregation ratio (isolate 2: 96 resistant:5 susceptible, $\chi^2_{13:3}$=13.21, P=≤0.001; isolate 10: 96 resistant:5 susceptible, $\chi^2_{13:3}$=13.21, P=≤0.001). This finding was validated by surveying disease responses from five F_3 families of this critical cross, all of which were resistant. Since all powdery mildew resistance genes that are located on chromosome 1A are dominant (*Pm3*, *Pm17* and *Pm25*; Hsam and Zeller 2002), we assume that chromosome 1A is the carrier of the dominant resistance gene and that the recessive gene is located elsewhere. Resistance gene *Pm17* was derived from rye cultivar Insave and is located on the short arm of rye chromosome arm 1R in a T1AL 1RS translocation. PCR analysis of a molecular marker that can be used as an indicator for the presence of *Pm17* (Mohler et al. 2001) conclusively demonstrated that line TA2682c does not carry this translocation (data not shown). The dominant gene is rather a member of the complex *Pm3* locus than *Pm25*, which originates from *T. monococcum* ssp. *aegilopoides*. We will be able to answer this question in future investigations when diagnostic DNA markers for *Pm3* and *Pm25* loci become available. The chromosomal location of the recessive resistance gene remains unknown since no other cross of CS monosomics and TA2682c deviated from the 13:3 ratio.

Development of a mapping population segregating for the recessive *Pm* resistance gene

For genetic mapping of the recessive resistance gene with molecular markers, a population segregating exclusively for the recessive resistance gene was produced. Susceptible F_2 plants were selfed under the assumption that a two-thirds of the plants were heterozygous for the recessive resistance gene locus. Resistance reactions of a random sample of 42 F_3 families (35–40 progeny of individual susceptible F_2 plants) were assessed individually on detached primary leaves following inoculation with Bgt isolate nos. 2, 9, 10, and 13. Twenty F_3 families segregated in their resistance response. This observed number deviates from the theoretically expected number of 28 segregating F_3 families (20 segregating F_3s:22 non-segregating F_3s, $\chi^2_{2:1}$= 6.60, P=0.04). The segregation for resistance to powdery mildew added up over the 20 families corresponded to the expected 1:3 ratio (184 resistant:554 susceptible, $\chi^2_{1:3}$=0.0054, P=0.94). Further evidence supporting the single-gene model was supplied from the observation that nearly all individual F_3 families (18/20) showed a 1:3 segregation. Twenty-eight homozygous-susceptible and 32 resistant F_3 plants were randomly chosen and selfed to form a mapping population consisting of 60 F_4 families, which is considered to be a sufficient number for estimating the extent of linkage between loci (Paterson 1996).

Molecular mapping of the recessive *Pm* resistance gene

To determine the chromosomal location of the recessive resistance gene, we assayed bulked segregants from contrasting F_3 phenotypes with a total of 176 *Eco*RI+ANN/*Mse*I+CNN AFLP primer combinations. Out of roughly 18,700 amplified marker loci, two AFLPs, namely *XE39M58-414* and *XE33M62-392*, were polymorphic between both the resistant and susceptible bulked segregants and the parental lines and, therefore, putatively linked to the recessive resistance gene. *XE39M58-414* was linked in repulsion (Fig. 1A), while *XE33M62-392* provided a marker in coupling phase. To test whether or not the selected AFLP markers show strong association with the recessive resistance gene, we used the DNA from individual plants of each F_3 bulk in AFLP reactions. Both AFLP markers were completely linked to the resistance gene within individuals from each bulk and, hence, were scored across the F_4 mapping population. The assignment of marker *XE39M58-414* amplified from the genome of CS and, allusively, the recessive resistance gene to wheat chromosome 7A was simply achieved by AFLP analysis of CS nullitetrasomics. The allocation of *Pm* resistance genes by means of nullitetrasomic analysis of repulsion phase AFLP markers obtained from experimental crosses involving CS has already been carried out for *Pm24* (Huang et al. 2000b) and *Pm1e* (Singrün et al. 2003). This result implies again that wheat chromosome 1A harbors the dominant resistance gene.

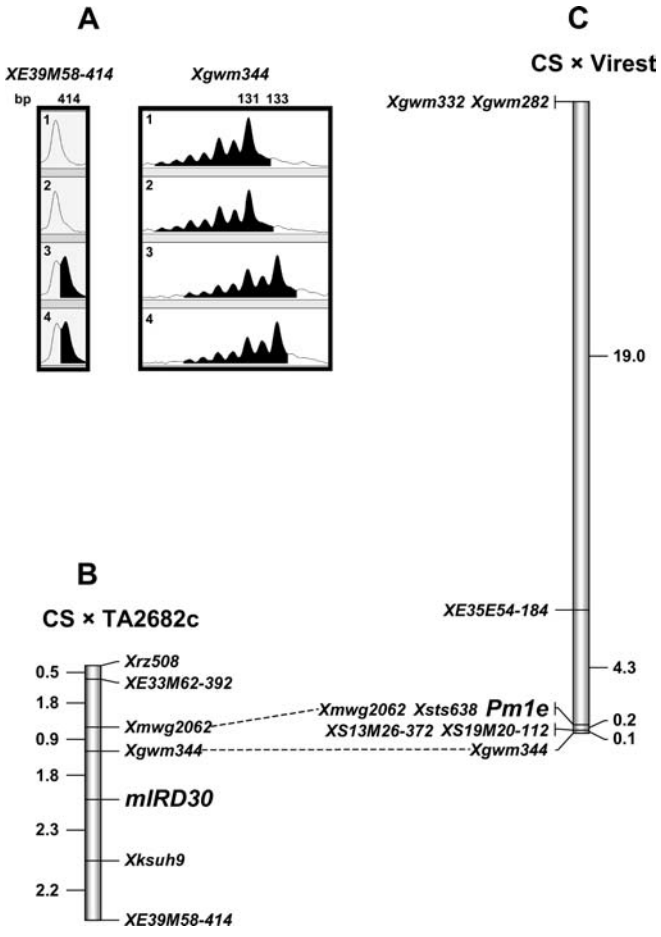

Fig. 1A–C Genetic mapping of the recessive resistance gene *mlRD30* from wheat line TA2682c. **A** Bulked segregant analysis of markers *XE39M58-414* and *Xgwm344*: *1* TA2682c, *2* resistant bulk, *3* susceptible bulk, *4* Chinese Spring (*CS*). Relevant peaks are marked in *black*. *Y-axis* shows the relative signal intensity of fluorescently labeled DNA fragments; *X-axis*, the size of DNA fragments in basepairs. **B** Linkage map around *mlRD30* from CS × TA2682c, **C** linkage map around *Pm1e* from CS × Virest. *Dashed lines* connect orthologous molecular marker loci between mapping populations

An inspection of seven SSR markers evenly distributed along wheat chromosome 7A refined the position of the recessive resistance gene to the terminal region of chromosome 7AL since *Xgwm344*-7AL was the only SSR locus displaying polymorphism between both the parental lines and the phenotypic bulks (Fig. 1A). *Xgwm344* dis- played allele sizes of 131 bp and 133 bp from resistant and susceptible DNA bulks, respectively. Four RFLP markers that had been previously mapped in the distal region of chromosome arm 7AL were surveyed on the parental Southern blots. Following the integration of segregation data from RFLP markers *Xrz508* from rice, *Xmwg2062* from barley, and *Xksuh9* from *Aegilops tauschii* and SSR marker *Xgwm344*, a genetic recombination map involving six molecular marker loci around the recessive resistance gene could be constructed (Fig. 1B). *Xrz508*, which was the most proximal marker locus to the recessive resistance gene, followed a codominant segregation, while *Xmwg2062* provided a marker in coupling phase. *Xksuh9* showed a *Dra*I restriction fragment in the susceptible DNA bulk that was absent in the resistant DNA bulk.

The recessive *Pm* resistance gene locus maps distal to *Pm1*

The location of the recessive powdery mildew resistance gene relative to *Pm1* was determined by connecting the genetic map from CS × TA2682c to that from CS × Virest (*Pm1e*) (Singrün et al. 2003), in which markers *Xmwg2062* and *Xsts638* were previously integrated. In the study of Neu et al. (2002), *Xmwg2062* mapped 0.4 cM proximal to *Pm1* in population Chancellor × Axminster/8*Chancellor and cosegregated in population Frisal × Sappo, while *Xsts638* showed no recombination to *Pm1* in both populations. For the map calculations, the number of the original marker set was reduced to encompass *Xmwg2062*, *Xsts638*, *Xgwm282*, *Xgwm332*, *Xgwm344* and dominant AFLP markers in the coupling phase only. Genetic mapping using this clearly arranged data set showed *Xmwg2062* and *Xsts638* to cosegregate with *Pm1e* but placed *Xgwm344* distally to the resistance gene (Fig. 1C). This is in conflict with the previous mapping result employing the full marker set (Singrün et al. 2003) but conforms the marker order from genetic map of CS × TA2682c (Fig. 1B). The observed disturbance in marker order of the original data set might be due to complications in positioning tightly linked coupling phase and repulsion phase markers as accurately as possible (Mester et al. 2003). Altogether, based on common markers *Xmwg2062* and *Xgwm344* between mapping populations CS × TA2682c and CS × Virest(*Pm1e*) it can be concluded that the recessive powdery mildew resistance gene in common wheat line TA2682c maps distal to *Pm1* on wheat chromosome 7AL.

The disease response pattern of the single-resistance gene line RD30, together with wheat lines possessing documented powdery mildew resistance genes were characterized using 11 differential Bgt isolates (Table 2). The specific resistance pattern of line RD30 was clearly distinguishable from those of wheat lines carrying other known genes in that it showed full resistance to all Bgt isolates. Powdery mildew resistance gene *Pm9* has been reported to be linked to *Pm1*, which is located on chromosome 7AL (McIntosh et al. 1998). Employing F_2 segregation analysis of Normandie × Federation hybrids, Schneider et al. (1991) found that this gene shows a recessive inheritance and is linked to *Pm1* with a map distance of 8.5 cM. Since cv. Normandie, which possesses gene combination *Pm1+2+9*, was susceptible to four Bgt isolates (Table 2), it can be ruled out that Normandie carries the same resistance allele as RD30. This finding was confirmed by testing cv. Pompe, which has gene combination *Pm1+9*; Pompe displayed the same reaction pattern as Axminster/8*Cc (*Pm1a*). An allelism test for *Pm9* is not feasible because, at

the present time, no wheat line that carries only *Pm9* is available. Therefore, the recessive gene from wheat line RD30 is tentatively designated *mlRD30*.

The recessive resistance gene *mlRD30* conferred full resistance at the seedling stage to all of the different Bgt isolates used in the present study. Therefore, introgression of *mlRD30* may be beneficial to future wheat breeding programs searching for resistance to powdery mildew. However, for a sustainable gene management to be achieved, sound decisions with respect to its diversification have to be made: it is anticipated that the combination of *mlRD30* with novel effective genes—for example, *Pm24* (Huang et al. 2000b) and *Pm5e* (Huang et al. 2003)—that have not yet been used extensively will provide a more durable protection than its combination with less effective genes—for example, *Pm2*, *Pm4b*, and *Pm8* (Felsenstein and Jaser 2000).

Acknowledgements The authors gratefully acknowledge Amalie Fiedler and Christina Hartmann for their excellent technical assistance and the anonymous referees for valuable suggestions.

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